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Money, schooling, and health: Mechanisms and causal evidence

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An association between higher educational attainment and better health status has been repeatedly reported in the literature. Similarly, thousands of studies have found a relationship between higher income and better health. However, whether these repeated observations amount to causality remains a challenge, not least because of the practical limitations of randomizing people to receive different amounts of money or schooling. In this essay, we review the potential causal mechanisms linking schooling and income to health, and discuss the twin challenges to causal inference in observational studies, in other words, reverse causation and omitted variable bias. We provide a survey of the empirical attempts to identify the causal effects of schooling and income on health, including natural experiments. There is evidence to suggest that schooling is causally related to improvements in health outcomes. Evidence also suggests that raising the incomes of the poor leads to improvement in their health outcomes. Much remains unknown beyond these crude findings, however; for example, what type of education matters for health, or whether there is a difference between the health impacts of temporary income shocks versus changes in long-term income.

Keywords: education; income; SES

Introduction

Both education and income appear to be robustly associated with health status. Whether these associations represent causation has proved harder to demonstrate because of the practical constraints on randomizing groups of people to receive different levels of money or schooling. This essay is organized into four sections. In the first section, we review the hypothesized mechanisms linking income and education to improved health outcomes. In Section 2, we discuss the twin problems of reverse causation and unobserved heterogeneity that bedevil observational studies of income/education and health. In Section 3, we survey the empirical studies that attempted to overcome problems of endogeneity, including quasi-experiments. The final section discusses what remains to be understood about the relationships between schooling/money and health, that is, what we need to know to translate knowledge into policy.

Mechanisms linking education and income to health

Income and health

If income is causally related to health (and that depends on whether one is convinced by the empirical evidence—see later section), the proposed mechanisms generally involve either access to material resources (e.g., the ability to purchase higher quality diets, or better housing), or access to symbolic resources (status and rank within one's community), or both. Broadly speaking, three different hypotheses can be spelled out regarding the relationship between income and health.[1]

First, the *absolute income* hypothesis posits that

$$h_i = f(y_i),\ f_i' > 0,\ f_i'' < 0, \qquad (1)$$

where h_i is an individual's level of well-being (e.g., years of life), and y_i refers to that individual's own level of income. The relationship between individual income and individual health is shown as a

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diminishing function of increasing levels of income because above the level where basic needs are met, added income has fewer health benefits. By contrast, the *relative income* hypothesis posits that

$$h_i = f(y_i - y_r), \tag{2}$$

where the term $(y_i - y_r)$ denotes the relative gap between an individual's income, y_i, and the income of some reference group, y_r. The reference population could be the income of co-workers, neighbors, or the national population. In this instance, there is no asymptote; the greater the gap, the poorer one's health.

The absolute income hypothesis is primarily identified with the so-called "materialist" theory[2] that attributes income effects on health as resulting from access to tangible resources such as food, clothing, and shelter; while the relative income hypothesis is primarily identified with "psycho-social" theory, which posits that the effects of income on health are mediated through symbolic resources such as status, prestige, and control. However, matching these hypotheses to one or the other theory is problematic and likely to be counterproductive. Both the absolute and relative income hypotheses are consistent with *either* neo-material or psychosocial explanations. Indeed, it is doubtful that an empirical study could ever succeed in teasing out neo-material from psychosocial processes because of colinearity and measurement error. It is difficult, if not impossible, to conceive of an experiment in which neo-material resources could be manipulated without also affecting psychosocial responses, and vice versa. Under the absolute income hypothesis, a given increase in income could improve health because it improves access to material resources but it simultaneously improves a person's sense of financial security. Conversely, under the relative income hypothesis, raising an individual's income relative to their reference group could improve health because it elevates their prestige but it also expands access to a broader range of goods and services that others cannot afford.[3]

The principal merit of distinguishing between the absolute and relative income hypotheses lies in the ability to make separate predictions about health effects even if the explanations are not obvious. This can be seen in the following thought experiment: If your current income is US$ 10,000 and everybody else's income in your community is US$ 20,000, what would happen to your health status if your neighbors' incomes were doubled but you were left with the same income (assuming equal purchasing power in the new scenario)? The absolute income hypothesis would predict that your health would remain unaffected. The relative income hypothesis would predict adverse health effects because the gap between your income and your reference group has been doubled. However, the reasons for this could reflect either psychosocial or material pathways. Your neighbors can now afford to purchase cell phones, an internet connection, obtain loans for a car or home, and so on. This could have an adverse impact on your health as a result of psychosocial effects of envy and frustration. At the same time, lack of access to goods and services that you cannot afford—but everybody else now can—could deleteriously affect your ability to participate and function within your community (a neo-material explanation). In practice, teasing out the absolute income effect from the relative income effect is tricky because of colinearity. Nevertheless, emerging empirical evidence suggests that absolute and relative income independently predict mortality, disability, and high-risk coping behaviors.

A few studies have now analyzed the related concept of *relative deprivation* (RD) using the Yitzhaki construct[4] based on the difference between own income y_i and mean income of those individuals j with higher incomes within the reference group of size N (weighted by the proportion of the reference group with income greater than i's)

$$\mathrm{RD}_i = \frac{1}{N} \sum_j (y_j - y_i) \; \forall y_j > y_i, \tag{3}$$

This relative deprivation construct focuses on the gap between ones own income and incomes of those richer than oneself, but ignores information on the magnitude of the income gap compared to those poorer than oneself. Eibner and Evans[5] find that relative deprivation compared to one's state-demographic reference group is associated with higher adult male mortality after controlling for own income. It is also associated with other adverse health outcomes such as seeking care for mental health problems, increased cigarette smoking as well as higher BMI, consistent with heightened stress which is one of the pathways by which deprivation could affect health outcomes.[5,6] However, these results are sensitive to the measure of relative income used, and the design does not rule out omitted

variables bias and reverse causality concerns discussed later. Further work in this vein would be valuable; for example, Kondo *et al*.[7] find that a similar relative deprivation measure is associated with higher disability incidence in a prospective cohort study of older Japanese adults.

Finally, yet a third formulation of the relationship between income and health is the *relative rank* hypothesis, which posits that an individual's level of health is determined by the relative position within a hierarchy that a given income confers on that individual. Related to the relative income hypothesis, evidence for the relevance of hierarchical rank on health derives from studies in nonhuman primates, for example, macaques and baboons, in the wild and in captivity. In both settings, higher-ranked animals have better health than do those who are lower on the dominance hierarchy. It is not simply that dominant individuals enjoy greater access to food and mates, however; adverse health effects of lower rank occur even when there are abundant resources. Rather, lower-ranked animals suffer a different set of slings and arrows of their subordinate status which appear to expose them to more stress.[8] Higher physiological cost of subordinate rank turns out to vary depending on the pattern of social organization in different species, the stability of the environment, and the temperament of the individual animal. For example, low-ranking individuals tend to be more stressed in stable hierarchies, whereas high-ranking individuals experience greater stress in unstable arrangements.

Complexities such as those described earlier raise questions about the relevance of evidence on dominance hierarchies in nonhuman primate species for human society. In addition, humans differ from other primates in having multiple bases of social ordering. There is not a single hierarchical order affecting a given individual; he or she may occupy different positions depending on the domain and reference group. Even within the domain of socioeconomic status, the various components (income, education, and occupation) are only moderately correlated with one another. Despite this, research using the MacArthur scale of subjective social status (SSS), on samples from a wide range of populations, has shown that individuals appear to have an overall sense of their relative position in the socioeconomic hierarchy and that this perception shows significant associations with health outcomes. The SSS scale asks individuals to place themselves on one of the rungs of a l0-rung ladder where the top of the ladder is occupied by individuals with the most money and education and the most prestigious jobs and the bottom by those with the least money and education and the worst jobs or no job at all. The higher people place themselves on the ladder, the better their health. Scores on the ladder have been linked to self-reported global health and disease cross-sectionally,[9,10] as well as to change in health over time.[11] Ladder scores also relate to biological indicators of stress arousal including elevated heart rate and blood pressure, greater abdominal fat deposition, and morning rise in cortisol, and reduced gray matter volume in the anterior cingulate portion of the brain which modulates stress response.[12–14] At an ecological level it has been linked to mortality rates.[15]

Many of the associations between ladder scores and health-related outcomes remain significant when adjusted for objective indicators of SES. Subjective status may be linked to health above and beyond objective status because it provides a more sensitive and complete measure of social status than do the traditional indicators. Alternatively (or in addition), it may be that the experience of lower status is itself distressing and the physiological responses associated with feelings of relatively low status may themselves be harmful. There is no research to date which allows us to test these competing possibilities. Intriguing studies in human populations have suggested possibilities of status effects. For example, Redelmeier and Singh[16] reported that, among those nominated for Academy Awards in acting, those who won the Oscar subsequently lived longer than those who did not. However, there is controversy over potential methodological flaws (see, e.g., Sylvestre *et al*.[17] and difficulties in distinguishing status effects from other risk protections that accrue to people with higher status).

Education and health

As in the case with income and health, there is consistent evidence linking more education with better health. The causal mechanisms underlying the link between schooling and health may operate through both material and psychosocial mechanisms. Education equips individuals with general as well as specific knowledge and skills that are useful for

prevention of disease. At the same time, higher educational attainment confers greater prestige and status within the community as well as serving as a credential for employment. Earning a degree increases one's chances for obtaining a job that pays well, has prestige, and exposes workers to fewer safety hazards. Data showing that the association of education and health is not perfectly linear (i.e., not every year of additional education contributes the same amount to better health) but is discontinuous at the times of degrees (e.g., 12 years, l6 years) suggests that a "sheepskin effect" may be responsible for some of the health benefits of education.

The causal inference problem

Although education and income both exhibit strong graded associations with health outcomes (mortality, morbidity, and health behaviors), these associations do not necessarily imply causality. Broadly speaking, there are two kinds of threats to causal inference: (a) reverse causation, whereby an observed association between money or schooling and health is explained by poor health status causing lower educational attainment or earnings, rather than the other way round; and (b) confounding of the association between money/schooling and health by unobserved third variables such as ability (IQ) or time preference.

Reverse causation

Reverse causation can arise even within panel designs in which SES measured at baseline predicts subsequent changes in health status. For example, suppose that income assessed in a panel of working-age adults is found to predict subsequent mortality risk (as has been reported in the Panel Study of Income Dynamics[18]), this longitudinal association does not necessarily demonstrate causality if the *initial* incomes of individuals were influenced by their health status during the time period prior to baseline. Even if we could measure and control for both health status and incomes during earlier time periods (i.e., a repeated measures design), causal inference remains elusive because of the likely reciprocal relations (simultaneity) between the two variables. This dynamic is illustrated in Figure 1.

Indeed some researchers assert that at older ages the association between income and health *mainly* reflects this type of reverse causation.[19] If so, increasing income of older people would not affect their health status. This may explain the paradoxical findings from analogies of health effects of the Social Security "notch" (see later section).

Reverse causation between income and health can arise for reasons other than illness impairing an individual's ability to be productive. For example, in the United States, lower incomes are strongly associated with overweight/obesity among adults. Closer inspection of this pattern reveals that it holds mainly for adult women, but not for men. The reason appears to be reverse causation operating through "fat bias" in society in relation to women, that is, overweight/obese women experience greater difficulty competing in the labor market and marriage market, leading to lower earnings, lower probability of getting married, and even lower spousal earnings. In a 15-year follow-up study of the Panel Study of

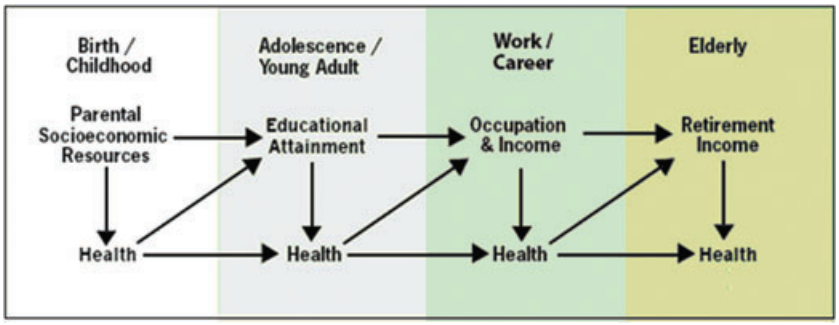

Figure 1. **The dynamic and reciprocal relationships between SES and health through the life-course. (*Source*: Adler, N.E., Stewart, J., and members of the MacArthur Network on SES and Health. 2008. *Reaching for a Healthier Life. Facts on Socioeconomic Status and Health in the U.S.* University of California, San Francisco. Accessed at: http://www.macses.ucsf.edu/News/Reaching%20for%20a%20Healthier%20Life.pdf)**

Income Dynamics employing the sibling fixed effects design, Glauber and Conley[20] examined differences in economic outcomes of siblings who were discordant with respect to body mass index at the beginning of follow-up. The sibling fixed effects design cancels out unobserved confounders such as early family circumstances. The authors found that among women, a 1% increase in BMI was associated with 0.6% lower family income, 1.1% lower spousal earnings, as well as 0.3% lower probability of getting married during follow-up. Notably, no associations were found between BMI and economic outcomes for men, suggesting that U.S. males do not pay the same penalty for being overweight, that is, reverse causation appears to be specific by gender and operates via a mechanism of societal prejudice against overweight women, rather than through any influence of obesity on the ability of women to be economically active.

It has often been claimed that education is less susceptible to reverse causation because most people have completed their schooling by the time they succumb to chronic diseases in adulthood. However, careful analyses of birth cohort data, such as the 1958 British Birth Cohort (the National Child Development Study, NCDS), reveal that chronic health conditions during childhood do indeed exert an adverse impact on educational attainment.[21] They examined the association of childrens' health with the number of "O-level" examinations they passed at age 16. These exams not only assess achievement but also affect one's chances for admission to university. Even after taking into account household and parental characteristics, each chronic condition reported at age 7 lead on average to 0.3 fewer O-level examinations passed. In short, chronic conditions during childhood—such as diabetes, ADHD, or mental health problems—probably led to children missing school which in turn, limited their achievement and future prospects.

Omitted variable bias

Both income and education are susceptible to confounding by unobserved third variables. For example, the relation between higher education and lower smoking status is often cited as an instance of the potential health benefits of schooling. However, when Farrell and Fuchs[22] examined the relationship between schooling and smoking within a community sample of adults who had completed from 12 to 18 years of education, they found that educational differences in smoking rates observed in adulthood (mid-20s) were already evident at age 17 when all of the subjects were still in the same grade. In other words, educational inequalities in smoking were evident even before schooling was actually completed. The authors suggest that a third variable such as time preference—as opposed to schooling *per se*—was responsible for the observed association between schooling and smoking prevalence. More recently, Fujiwara and Kawachi[23] conducted an analysis of twins in the National Survey of Midlife Development in the United States (MIDUS) and found that among dizygotic male twins, each additional year of schooling lowered the prevalence of smoking by 32% (odds ratio = 0.68, 95% confidence interval: 0.48–0.97) in fixed effects analysis. However, an association between schooling and smoking status was not found among monozygotic twin pairs, suggesting that the relationship between education and tobacco use may be confounded by unobserved inherited characteristics.

When we turn to the relationship between income and health, it has again been suggested that the association reflects confounding by underlying (and perhaps inherited) ability, as measured by IQ—the so-called "Bell Curve Hypothesis" (see Gottfredson 2004[24]). In other words, smarter people are able to earn more money *and* look after their own health. However, Link *et al*.[25] examined two longitudinal data sets (the Wisconsin Longitudinal Study and the Health and Retirement Survey) that included measures of both cognitive ability and income, and found that controlling for IQ did not remove the effect of income on health outcomes (mortality and self-rated health), whereas the association between intelligence and health disappeared once income and education were held constant. A natural experiment is also provided by children who are adopted by parents with different levels of income. Because adopting parents usually do not get to pick and choose who to adopt based on their background socioeconomic circumstances, the adoption process acts like a lottery that randomly assigns children to households with different levels of income. If IQ completely explains the association between income and health, then the adoption process should eliminate any association between the health of children and the incomes of the households that they are sent to. However, an analysis of adopted children in the

National Health Interview Survey failed to corroborate this hypothesis. The gradient between higher incomes and better higher status continues to be observed among adopted children, and is of a similar magnitude compared with children raised by their biological parents.[26]

Identification strategies for assessing causality

Broadly speaking, there are three sets of strategies for identifying the causal effect of income and education on health outcomes. They are—in descending order of strength of causal inference permitted—(i) experiments in which income or education is directly manipulated, (ii) quasi-experiments in which the researcher can take advantage of a naturally observed exogenous change in either income or education, and (iii) longitudinal, observational studies with careful control for confounding variables.

Income and health

Experiments

The New Jersey–Pennsylvania Negative Income Tax Experiment[27] assigned 725 eligible families to one of eight negative tax plans (which were combinations of guarantee levels and tax rates), and compared their outcomes over a 3-year period to 632 control families. The health outcomes studied included the number and type of chronic illnesses, the number of days spent in hospital, the number of days of work lost due to illness, as well as physician visits. Overall, the study found little evidence of any effect of payments on the measured health outcomes.

The PROGRESA/Oportunidades program in Mexico is a conditional cash transfer intervention that was initially phased-in using a randomized community cluster experimental design. Cash payments to poor families are tied to specific incentives to invest in the education, nutrition, and health of children. Gertler[28] found that eligible children in experimental communities had less parent-reported illness, less anemia, and improved height growth compared to control communities during the first 2 years of the program. It is unclear, however, to what extent these results are driven by the cash income as compared to other components of the program such as required well child care and health education sessions. Fernald *et al.*[29] used a quasi-experimental approach to attempt to isolate the effect of the cash income on young children, finding that income was indeed associated with higher child height-for-age and less stunting, as well as improved cognitive outcomes and motor development. A similar quasi-experimental design although found that among adults, higher Oportunidades cash transfers were associated with increased obesity and hypertension, perhaps reflecting greater financial ability to speed the nutritional transition toward overnutrition.[30] Subsequent cash transfer programs elsewhere have yielded further experimental evidence of health effects. Of particular interest for isolating the role of income is an *unconditional* cash transfer program in Ecuador that Paxson and Schady[31] find improves child anemia and cognitive outcomes but has no significant effect on child growth.

Quasi-experiments

Winnings from lotteries have been proposed as quasi-experiments. Lindahl[32] examined the longitudinal effects of lottery payments on health status in three waves of the Swedish Levels of Living Surveys (1968, 1974, and 1981) in which respondents were asked about amounts of money earned from betting or playing the lottery. Levels of family income were then instrumented using the amount of lottery payments, that is, the effect of changes in health were examined over the range of exogenous variation induced by the lottery winnings. The results suggested that each 10% increase in income was associated with a statistically significant 0.01–0.02 standard deviation increase in an index of health status, or an increase in life expectancy by between 5 and 8 weeks. Although a 5- to 8-week gain in life expectancy may appear trivial, it is comparable in magnitude to the gains in life expectancy estimated in the United States for counseling to stop smoking (see Bunker *et al.*[33]). The analysis was limited, by the fact that the survey did not distinguish between people who played the lottery versus those who never played the lottery. However, because lottery players tend to have lower educational attainment than nonplayers, the IV estimates are likely to be lower bounds of the effect of income on health, that is, people who reported "no lottery earnings" were a combination of people who regularly played the lottery but never won prizes *plus* those who never bet (and were likely to be more educated and hence healthier on average).

Ettner[34] carried out an IV analysis of income and health using data from the National Survey of Families and Households, the Survey of Income and Program Participation, and the National Health Interview Survey. She examined a range of health outcomes available in these data sets including self-assessed health status, functional limitations, depressive symptoms, and drinking behavior. Both OLS and IV estimates suggested positive associations between higher incomes and improved physical and mental health outcomes. However, the choice of instruments in this analysis is debatable. Ettner used as instruments (a) determinants of an individual's wage rate (the respondent's work experience and the state unemployment rate) and (b) determinants of the individual's non-earnings income (parental education as a proxy for bequests made to children, and spousal level of education and work experience). To the extent that arguments can be mounted that each of these variables exert an independent influence on health (and it seems, they can), these instruments fail to convince.

A different kind of natural experiment occurred with the so-called "Social Security notch," in which a change in the Social Security legislation resulted in higher benefits to individuals born before January 1, 1917, compared to those born afterward.[35] The authors compared mortality rates after age 65 for males born in the second half of 1916 and the first half of 1917, and found that the higher income group paradoxically experienced a *higher* mortality rate. The younger cohort (who received less money) appeared to increase their postretirement work effort, leading the authors to conclude that higher cash payments had a deleterious effect on the work incentive and health of the older cohort by encouraging them to stop working.

A different result was found in relation to children's health in a much lower income setting. Case[36] examined the effects of a large exogenous increase in income associated with the South African state pension system. In that country, elderly Black and Colored citizens who did not pay into the pension system (and who did not anticipate receiving payments) ended up receiving large pensions—roughly twice the median Black income per capita. Employing a difference-in-difference (DD) design, the author found that the health of household members who lived with pensioners (and pooled their incomes) improved significantly following the increase in income compared with members of households who did not live with pensioners, or who lived with pensioners but did not choose to pool incomes. Improvements were noted for children's height and nutritional status, as well as the self-reported health and depressive symptoms of adults sharing the payments with the elderly pensioners. Interestingly, in the minority of households that did not pool money, the health benefits of the pension seemed to be isolated to the pension recipient, and not to their extended family. The self-reported health status of pension recipients in these households (adjusted for age, race, and gender) was a full step better (e.g., moving from "average" to "good") than that of other household members. Duflo[37] similarly found that pension income appeared to improve child anthropometric status, although effects were concentrated only among girls, and only resulted from pensions given to women (grandmothers) and not from pensions to men.

In rural North Carolina, Costello *et al.*[38] observed a natural experiment in which a casino opened on an Indian reservation adjoining a community where they happened to be conducting annual psychiatric surveys among disadvantaged children aged 9–13 years. The casino opening happened halfway through the 8-year study, and resulted in an income supplement of about US$ 6,000 per year to American Indian families in the sample (about one quarter of the sample). Among families who received payments, 14% moved out of poverty resulting in an improvement in child psychiatric symptoms over time such that although they began with a level of psychopathology comparable to poor children, they eventually moved to the level typical of a non-poor child. Some questions remain, however, given that the main findings were not presented by intention-to-treat, and the direct effect of income on health was not reported (the authors presented the effect of change in poverty status instead).

Of direct relevance to U.S. policy debates are quasi-experimental analyses of the earned income tax credit (EITC), based on analyzing state and time variation in benefit generosity across demographic groups. Among adults, Schmeiser[39] suggests a potential negative effect of added income, finding that EITC income significantly raises female obesity (with no effects for men) in the low-income target population. The point estimates suggest that EITC earnings can explain about one quarter of

increased obesity prevalence in this group from 1990 to 2002, although the confidence intervals are reasonably large.

Longitudinal, observational studies

Smith[40] reported in the Health and Retirement Survey that there was no direct causal effect of income on health among respondents in this survey after controlling for prior health status; instead, he argued that the predominant cause of the income–health association in these older adults was due to health shocks leading to income loss. A similar conclusion was reached by Adams *et al*.[19] who conducted tests of Granger causality in the Asset and Health Dynamics of the Oldest Old (AHEAD) Panel. (However, this conclusion is debatable, because their tables of results appear to suggest much stronger evidence than they reported.) Smith[41] further argues that the Panel Study of Income Dynamics data supports the hypothesis that one's financial resources play a minor role in shaping health outcomes over the life course.

A number of longitudinal studies on income gradients have emphasized the role of early childhood SES in shaping later income–health gradients, although the precise role of income is hard to assess. Influential work by Case *et al*.[26] showed evidence of the early childhood origins of the later life income–health gradient, and argued that parental permanent income may have an important impact on health across the life course. Currie and Stabile[42] showed similar patterns in a Canadian panel, in which children are fully insured, suggesting that the income gradient in child health in the United States is likely unrelated to medical care access, but rather reflects the greater number and intensity of health shocks experienced by the lower income children. Case *et al*.[43] provide evidence for a similar story when comparing with the United Kingdom. Propper *et al*.[44] in turn find that the UK childhood gradient can be largely accounted for by the mother's health status, especially mental health, and similar results are found in Australia by Khanam *et al*.[45] The direction of causality between maternal health and income is not addressed in this work, but following the above pattern of findings, there may well be intergenerational origins of this as well, as found in Currie and Moretti.[46]

In summary, the causal evidence linking income to health remains mixed. Many of the studies that support a causal relationship are limited by methodological flaws, while the negative studies do not necessarily rule out a causal relationship. For example, several of the null studies tend to be in older populations (HRS, AHEAD, Social Security notch), which suggests that boosting incomes in this age group may not affect their health outcomes. However, this does not rule out a causal effect of income at younger ages. Indeed, in the HRS analyses[40] family income during childhood remained a significant predictor of adult health even after controlling for prior health status. And because boosting early childhood income means boosting the incomes of the parents of children, the policy implications may be the same, that is, raise the incomes of poor adults in order to improve the health of their children. It is also likely that transitory income shocks have different effects on health than do gradients and changes in permanent income. Indeed, the literature on the aggregate health effects of macroeconomic shocks suggests that many dimensions of health may temporarily improve during recessions (see, e.g., Ruhm, 2007[47]; Granados[48] with accompanying commentaries), although long-term secular income growth is strongly positively correlated with health. Similarly, while in developed countries such as the United States there is little evidence that short-term income improvements will have large health benefits, long-term investments in raising incomes of lower SES groups could indeed have larger benefits cumulating over generations.

Education and health

Experiments

The High Scope/Perry Preschool Program has been cited extensively in the literature.[49] The study was a small ($N = 123$) randomized experiment of an intensive, high-quality pre-kindergarten education program delivered to children born in poverty. Follow-up of these individuals up to 37 years later found substantial benefits in a broad range of domains including readiness for school, subsequent educational success (high school and college graduation), earnings, and reduced number of criminal arrests throughout life. Despite these benefits, however, the participants in the treatment group did not exhibit any overall advantage in physical health outcomes by age 40 years compared to the control group.[49] We are unaware of randomized experiments of education at older ages.

Quasi-experiments

The Head Start program was established in 1964 as part of the War on Poverty, and provided funding and technical assistance to 300 of the poorest counties in the United States. The intervention included not only educational components, but also health services (e.g., screening for anemia and immunizations), nutritional programs, parenting skills, and even a jobs program. Thus, the effects of the program cannot be interpreted as the pure effects of early childhood education alone. Ludwig and Miller[50] applied a regression discontinuity design to county-level data, and found that 10 years after the roll-out of the program (1973–1983), a large reduction in child mortality rates was evident in vital statistics data, as well as mortality from specific causes such as asthma, anemia, infections, and diabetes.

Another widely cited natural experiment is provided by state-level variations in compulsory schooling laws. Historically, the number of years a child must remain in school in the United States has been determined by state laws. These requirements have varied historically between states, and they changed repeatedly in different states during the first half of the 20th century. States typically extended their mandatory schooling by either lowering the age at which children had to begin attending school, or by raising the age at which they could drop out or obtain a work permit. Leveraging off these "natural policy experiments," Lleras-Muney[51] carried out an instrumental variable analysis of schooling and mortality using the 1960, 1970, and 1980 U.S. censuses (linked to vital records) as well as the National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study, 1992. Her results suggested that education has a substantial impact on lowering the risk of mortality—each additional year of education lowers the probability of dying in the next 10 years by 3–6 percentage points. This study has been widely cited, and has also attracted other researchers reanalyzing the same data. Mazumder[52] has argued that the Lleras-Muney's results are fragile and have been too readily accepted. His arguments include: (1) when state-specific linear cohort trends are added to the models, the point estimates drop and become insignificant; this is a common robustness test, and the fact that the standard errors do not substantially increase suggests that this is a reasonable test and a major concern; and (2) schools with increased compulsory schooling laws may also have experienced other concurrent changes, such as changes in nutrition and smallpox vaccination programs. Other papers have used similar compulsory schooling laws to estimate education effects, with mixed results: McCrary and Royer[53] found no effect on infant health, but Glymour *et al*.[54] examined the association between schooling and cognitive functioning within the Health and Retirement Survey and found large and statistically significant effects of education on memory tests.

Currie and Moretti[46] took a different approach, exploiting local college openings during the 1960s and 1970s as an instrument for female college attendance, finding various health effects including improved birth-weight and reduced smoking.

Longitudinal, observational studies

As noted earlier Link *et al*.[25] sought to test the extent to which the relationship between income, education, and health outcomes are confounded by underlying ability, or intelligence. They examined two panel data sets—the Wisconsin Longitudinal Study and the Health and Retirement Survey—with information on adult SES, IQ, and health (mortality and self-rated health). They found that both higher education and incomes are robustly associated with better health outcomes even after controlling for intelligence. By contrast, the apparent association of intelligence with health outcomes disappeared after taking account of education and income. The authors cautiously conclude that their findings "are inconsistent with the claim that intelligence is the elusive fundamental cause of health disparities, and instead supports the idea that the flexible resources people actively use to gain a health advantage are the SES-related resources of knowledge, money, power, prestige, and beneficial social connections" (Link *et al*. 2008,[25] p. 72).

To summarize, there is evidence suggesting that the association between schooling and improved health outcomes is causal. There is also evidence that raising the incomes of the poor improves their health outcomes. At the same time, a note of caution is warranted, because not every association that has been reported between schooling and health outcomes is likely to be causal. As discussed earlier, the association between schooling and smoking is more likely to reflect confounding by third variables (such as time preference). In other words, more specificity

is required in pinpointing the exact types of health outcomes that are likely to be causally influenced by schooling.

What we do not know and what policy makers need to know

Beyond strengthening causal inference through the use of more rigorous study designs, there is much that we still do not know about the links between income, education, and health. For income, unanswered questions include: (a) distinguishing the effects of temporary income (income shocks) from the effects of permanent income. Income shocks may be associated with more harmful behaviors in the short term (e.g., cigarettes and booze become more affordable), whereas increases in permanent income may improve a person's prospects for the future and lead to increased incentives to invest in their longevity; (b) pinpointing the stage of the life course at which income matters most for health. Increasing evidence points to the importance of family income during early childhood as a determinant of health in later life, whereas the effects of adult income on adult health (especially at older ages) seem to be much weaker; and (c) clarifying whether income matters more in particular social contexts—for example, does income poverty have less of an impact on health in societies with strong safety nets and welfare state provisions (e.g., free health care, good quality public schools) that make income less salient for the participation of its citizens in the life of the community?

When we turn to education, many unanswered questions similarly remain. For example, what types of skills and knowledge are relevant for specific health outcomes? Are there critical periods in the life course during which education has a greater impact on the trajectory of an individual's health than at other times—for example, during pre-school or adolescence or college? Are the health benefits of an additional year of schooling constant no matter what the baseline level of education? Does it matter what kind of education you get—technical/vocational or liberal arts? Does the quality of education matter, and if so, which aspects of quality?

For both income and education, we need to get more specific about the relationships to different health outcomes. For example even if the association between income and obesity reflects reverse causation (for adult women), we cannot generalize this conclusion to other outcomes such as physical activity, and diet, both of which are linked to income and obesity. Likewise, even if the association between adult income and adult overweight/obesity is not causal, the relationship between parental income and childhood overweight/obesity may still be causal. And so on.

Over and above the potential main effects of education and income on health, we need also to understand whether there are potential spillover effects (positive externalities) of both on the health of others. Several types of spillovers have been reported in the case of education, including the impact of maternal education on improvements in child health; the influence of children's education on parental smoking quit rates; and the impact of own education on spousal health (although in the latter instance, there is a potential gender interaction—women married to more educated husbands tend to benefit, whereas men married to more educated women have been observed to suffer more heart attacks in the Framingham Heart Study![55]). To the extent that education has spillover effects, there will be market failure—that is, private decisions to invest in schooling will not have incorporated these benefits—thus justifying state intervention to strengthen schooling.

Finally, when transported to the policy realm, health researchers need to be reminded to distinguish between the effects of income from the effects of education—as we have done throughout this paper. Although these two variables tend to be lumped together or even used interchangeably as aspects of SES (along with occupation), they are completely different interventions from a policy standpoint. Thus, incomes can be raised relatively quickly with a change in tax and transfer policy, whereas investing in education will likely require a generation before the health benefits can be reaped. That said, we understand relatively little about the potential interactions between education and income—for example, whether money is more effectively translated into health gains if the recipient is more educated; or whether it is even feasible to improve health by income transfers alone because permanent income appears to be more powerful.

In summary, the identification of causality has proved challenging in studies linking education and income to health. The stakes are high, because if the

association between income/education and health turn out not to be causal, then policies should be more usefully channeled toward interventions that directly improve health. If on the other hand, income and education are causally related to health, then any policy that affects either the levels of income (e.g., tax and welfare policies) or schooling need to factor in their potential consequences for population health and well-being. A better understanding of the sorts of questions raised in this essay remains the task for the next generation of research on SES and health.

Conflicts of interest

The authors declare no conflicts of interest.

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